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Swine Influenza A at Fort Dix, New Jersey (January–February 1976). IV. Summary and Speculation

Franklin H. Top, Jr. and Philip K. Russell

From the Department of Virus Diseases and the Division of Communicable Disease and Immunology, Walter Reed Army Institute of Research, Washington, D.C.

Influenza A/New Jersey/76 virus was detected at Fort Dix from January 19 through February 9, 1976 and infected at least 230 military personnel. Thirteen hospital admissions for acute respiratory disease were associated with influenza A/New Jersey infection, and additional members of index training companies may have been hospitalized with influenza A New Jersey. This virus was likely introduced into the reception center by an incoming trainee. Although our studies could not eliminate the possibility that influenza A/New Jersey strains are inherently less transmissible in humans than H3N2 viruses, the simultaneous transmission of influenza A/Victoria/75 virus and the unusual environment in basic combat training may explain why influenza A/New Jersey did not spread significantly outside of this training population.

Initial reports and the preceding papers [1–5] have described various features of the outbreak of influenza A/New Jersey/76 at Fort Dix, N. J. Here we summarize the accumulated information and address questions not fully answerable by data from the retrospective epidemiologic studies without additional assumptions.

Observations on the clinical manifestations of influenza due to the A/New Jersey virus were limited to 13 persons hospitalized at Fort Dix whose infections were confirmed by viral isolation or serologic studies [3]. One patient collapsed while on a training march and died; postmortem findings were typical of a severe viral pneumonia and pulmonary edema. Four of the remaining 12 influenza A/New Jersey patients had X-ray evidence of pneumonia; in three of these patients, polymorphonuclear leukocytosis and respiratory tract cultures yielding group A *Streptococcus* or *Haemophilus influenzae* suggested a concomitant bacterial infection. The proportion of influenza patients with pneumonia was higher than that observed in other reports [6]; however, the number of patients was small. Toms et al. [7] found influenza A/New Jersey/8/76 virus to be more pneumotropic in ferrets than strains of Asian or Hong Kong influenza virus. Otherwise, the disease seen in the 12 Fort Dix patients was similar to that described with previous influenza A strains [8] and more severe than that observed in the six volunteers inoculated with egg-passaged influenza A/New Jersey/8/76 virus [9]. Hospital admissions for acute respiratory disease (ARD) were proportionally greater in trainees with titers of antibody to influenza A/Mayo Clinic antigen of ≥1:20 than in trainees without antibody in five of six index platoons studied [4]; this finding suggests that more than the 13 patients described were hospitalized with influenza A/New Jersey infections.

The epidemic of influenza A/New Jersey at Fort Dix was brief, lasting from January 19 through February 9, 1976 [5]. Influenza A/New Jersey strains were not isolated after February 14 despite intensive sampling at Fort Dix [3] and extensive surveillance in surrounding townships by the New Jersey State Board of Health [1]. Transmission of influenza A/New Jersey virus (as estimated by prevalence of HAI antibody to A/Mayo Clinic/103/74 [HswN1] antigen at a titer of ≥1:20) occurred in six companies in basic combat training (BCT) which began training on January 12, 19, or 26, 1976. By a conservative estimate, 230 trainees in these six companies were infected with influenza A/New Jersey virus [5]. Transmission of influenza A/New Jersey virus also was documented in three additional BCT companies (D5, A5, and B7) and an advanced individual training (AIT) company. Also, some

Please address requests for reprints to Dr. Franklin H. Top, Jr., Division of Communicable Disease and Immunology, Walter Reed Army Institute of Research, Washington, D.C. 20012.

BCT and medical personnel in contact with recruits may have had influenza A/New Jersey virus infections. However, transmission of influenza A/New Jersey virus outside of BCT units was not extensive, and the virus was not detected in the civilian population at Fort Dix.

There are two additional questions that cannot be completely answered by the retrospective studies at Fort Dix without further assumptions but which warrant discussion because they pertain to the human transmissibility of the influenza A/New Jersey strain recognized at Fort Dix. How was the influenza A/New Jersey virus introduced into Fort Dix? Why did transmission of the influenza A/New Jersey virus stop at Fort Dix?

Introduction of influenza A/New Jersey virus into Fort Dix. It seems most probable that the virus was introduced through the reception center at the post by an incoming trainee. Although definitive proof of this hypothesis is lacking (and indeed the virus was not isolated from reception center trainees or personnel), there is considerable data consistent with it. The earliest influenza A/New Jersey cases hospitalized were BCT trainees admitted on January 19 and 20; these trainees had not yet begun training or were in the first week of training. However, case finding was biased toward recruits in the reception center and first four weeks of training so that we would not have detected cases by serologic study in trainees beyond the fourth week of BCT or in other military personnel at Fort Dix if influenza A/New Jersey occurred in these units.

There was no evidence of influenza A/New Jersey virus infection in the two companies sampled (B4 and C7) that began BCT on January 5 immediately upon return after Christmas leave [5]; trainees in these two companies were in the reception center immediately prior to the holiday leave period. However, transmission of influenza A/New Jersey virus (as estimated by prevalence of HAI antibody at a titer of $\geq$1:20) occurred in six of 14 companies that began BCT on January 12, 19, or 26. Two of four companies beginning training on January 12, two of five beginning on January 19, and two of five beginning on January 26 had a >10% prevalence of A/Mayo Clinic antibody. Most members of the cohort beginning training on January 12 would have passed through the reception center during the week beginning January 5, the first week after Christmas recess. The greatest transmission of influenza A/New Jersey virus then was found in the first three weekly cohorts of BCT trainees to enter the reception center after the Christmas holiday.

Rates of hospitalization for ARD began to rise in BCT trainees the week beginning January 18 when the initial influenza A/New Jersey cases were hospitalized. ARD rates were much higher in trainees in the reception center, those who had not begun training, and those in the first week of BCT than in more seasoned trainees; from January 18 through February 14, 42% of total recruit ARD admissions were trainees in this earliest part of training (J. Bartley, personal communication). Excluding identified cases, 72 patients with ARD were admitted from six index companies with 1,129 trainees (E6, C2, C4, E1, D6, and A6) from January 12 through February 1; 39% of the 31 of these patients studied serologically had antibody titers consistent with influenza A/New Jersey infection. Therefore, a large proportion of ARD hospitalizations in the first two weeks of the influenza epidemic occurred in trainees in the reception center and early weeks of training, and a large proportion of these hospitalizations were associated with influenza A/New Jersey infection.

After platoons leave the reception center and enter the environment of their new training company, BCT trainees are quite isolated from other military units on the post. Contact with other BCT companies, even those in the same week of training, is uncommon. Trainees in the first three weeks of training do not have liberty to leave the company environment individually except for medical care. Aside from the medical system, the only common experience of members of the six BCT companies with a high prevalence of A/Mayo Clinic antibody was their stay in the reception center in the weeks beginning January 5, 12, or 19. Therefore, it seems most probable that influenza A/New Jersey virus was introduced into BCT companies by members who were infected within the reception center, although we cannot eliminate the alternative possibility that virus was introduced by infection of trainees through several medical dispensaries ear-

ly in training. The first hypothesis would imply that infection was likely to have been introduced by an incoming trainee.

Why did influenza A/New Jersey transmission stop? The critical question remaining is why influenza A/New Jersey virus was no longer identified at Fort Dix after February 14, less than one month after it was first detected. In view of the competitive advantage afforded to the A/New Jersey virus by the near universal lack of antibody to Hsw and N1 antigens in the young military population, why did influenza A/Victoria virus prevail? It is possible that the A/New Jersey virus was inherently less transmissible than the A/Victoria virus. No studies of the transmissibility of the A/New Jersey virus in humans have been reported. However, Toms et al. consider that its transmissibility in ferrets might be similar to that of influenza A/Hong Kong (H3N2) strains [7]. Four to seven generations of influenza A/New Jersey virus must have been transmitted in Fort Dix trainees, since it caused hospitalizations over a 22-day period. Also, it infected a large proportion (10%–45%) of trainees in three companies. However, only 0–18% of trainees in four other index companies had influenza A/New Jersey infection. The infection rate with this virus was lower at Fort Dix than the 40%–60% infection rates described in certain closed military populations with influenza A/Hong Kong virus in 1968 [10–12]. Unfortunately, rates of infection with influenza A/Victoria virus could not be determined from the retrospective serum survey because of the high prevalence of A/Victoria antibody in incoming recruits and A/Victoria antibody responses induced by immunization of trainees with H3 antigens. Although the A/New Jersey virus may have been inherently less transmissible in humans, the unique environment in BCT and the unprecedented simultaneous presence of two radically different influenza A viruses on the post may have played an important role in inhibiting its spread. It is appropriate to examine how these factors may have impeded the transmission of A/New Jersey virus in the reception center, in BCT companies, and in the remaining Fort Dix population.

If one accepts the premise that influenza A/New Jersey virus was introduced into the reception center by an incoming trainee, the virus probably persisted from the week beginning January 5 through that beginning January 19 in order to account for infection of the three cohorts beginning training on January 12, 19, and 26. Since little influenza A/New Jersey activity was detected elsewhere in the United States in 1976, most probably only one or, at most, a few trainees introduced the virus into the reception center. However, because influenza A/Victoria was widely prevalent in the civilian population from January through March, this strain may have been introduced frequently by new trainees who were infected prior to arrival on the post. The short stay in the reception center (generally three days) would preclude significant amplification of the number of trainees shedding either influenza virus strain there; any inherent advantage in transmissibility of one influenza virus over another could hardly be expressed in one generation. Therefore, the relative proportion of incoming trainees arriving infected with a given strain, together with their immune status, would be the most important factors in determining the level of transmission of both influenza strains in the reception center. The continual introduction of A/Victoria virus into the reception center in contrast to the limited introduction of A/New Jersey virus may have resulted in a gradually decreasing possibility of individual exposure to A/New Jersey virus and a greater possibility of exposure to A/Victoria virus. Thus, A/New Jersey virus may have been overwhelmed in the reception center by the continual introduction of influenza A/Victoria strains.

Once BCT units left the reception center, they were relatively isolated at least until the fourth training week. The limited contact between trainees of different companies reduced possibilities of viral transmission between BCT units. By the time trainees of index companies were free to move about the post, influenza A/New Jersey virus was no longer present in these companies. Possible routes of transmission between BCT companies were limited to exposure of cadre to infected cadre of different units or exposure of trainees in one company to infected trainees of another in dispensaries or the hospital ARD wards. Past epidemics of ARD due to adenovirus in the second and third weeks of BCT during

January and February at Fort Dix provide evidence that isolation of BCT companies is not complete during the early training weeks [13]. However, the relative isolation of trainees at the very time influenza A/New Jersey was present in their units surely must have hindered the transmission of the new virus to other groups.

Influenza A/New Jersey was, of course, detected in units other than recently formed BCT companies. Two patients (V1 and V2) were in units that had post liberty. Some BCT cadre and medical personnel in contact with recruits may have had influenza A/New Jersey infection and could have disseminated the virus throughout the post. Why then did not influenza A/New Jersey spread more extensively at Fort Dix? Possibly antibody to influenza Hsw or N1 antigen induced by previous influenza A0 and A1 infections or by immunization with these and A/swine antigens reduced infection or transmission of the A/New Jersey virus by older persons. Possibly the extensive transmission of influenza A/Victoria virus in Fort Dix limited transmission of A/New Jersey virus. We are not aware of studies of the effect of recent or intercurrent infection by one influenza A virus on human infection or transmissibility of an influenza A strain with unrelated surface antigens. However, Murphy et al. did show that infection with influenza A/Hong Kong/68-*ts*-1[E] virus did not interfere with infection or shedding of parainfluenza type 1 virus by volunteers challenged seven days later [14]. Finally, to return to the opening theme of this section, the influenza A/New Jersey virus at Fort Dix may not have been as transmissible in humans as was the A/Victoria virus.

The limited human transmission from the sporadic cases of influenza A/swine encountered in the 1976–1977 winter season supports the latter hypothesis; data from epidemiologic studies at Fort Dix certainly do not refute it. However, it is important to realize that the environment in the reception center and training brigade at Fort Dix, together with the high-level, simultaneous transmission of influenza A/Victoria virus, might have significantly inhibited the transmission of a fully virulent and transmissible influenza A strain. Whereas the simultaneous occurrence of two radically different influenza A strains may permit the emergence of a natural recombinant with the human virulence of an established strain and surface antigens of the new strain, paradoxically, transmission of the established strain might inhibit spread of the new strain. The rapid disappearance of the influenza A/New Jersey strain at Fort Dix prohibited prospective studies which may have shed considerable light on the interactions between two radically different influenza A viruses infecting humans at the same place and time.

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